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Research Article

A cross sectional study on correlation between lipid profile and severity of liver disease

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ABSTRACT

Objective: To determine the correlation between lipid profile and severity of liver disease in cases presenting tertiary care Hospital, Bahawalpur.

Material and methods: This cross sectional study was conducted at Department of Gastroenterology & Hepatology, Bahawal Victoria Hospital, Bahawalpur from February 2018 to August 2018 over the period of 6 months. Total 123 patients with age ≥18 years either male or female with chronic liver disease or documented case of chronic liver disease irrespective of aetiology were included in the study. Correlation between lipid profile and child pugh class was studied.

Results: Mean age of the patients was 38.01 ± 14.68 years. Out of 123 patients of chronic liver disease, male patients were 104 (85%) and female patients were 19 (15%). Total 24 (20%), 47 (38%) and 52 (42%) patients belonged child pugh class A, B and C. In childpugh class A, B and C, Mean Total cholesterol was 139.08±40.16 mg/dl, 141.30±51.54 mg/dl and 105.81±40.26 mg/dl respectively. Mean LDL was 84.29±35.16 mg/dl, 88.55±48.13 mg/dl, 76.67±79.19 mg/dl respectively in child pugh class A, B and C. Mean HDL was 34.42±14.67 mg/dl, 29.81±12.82 mg/dl and 21.23±11.73 mg/dl in child pugh class A, B and C. Mean TG was 109.17±64.04 mg/dl, 116.32±54.99 mg/dl and 90.06±53.35 mg/dl respectively in child pugh class A, B and C.

Conclusion: Results of present study showed a higher percentage of male patients as compared female suffering with chronic liver disease. Most of the patients belonged to age group 51-60 years. Negative correlation between lipid profile and child pugh class was found.

keywords. Cirrhosis, lipoproteins, Chronic Liver Disease, CVD

INTRODUCTION

Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury, that leads to portal hypertension and end stage liver disease.[1] Autopsy studies done globally showed prevalence of cirrhosis ranging from 4.5 to 9.5% of the general population. Hence more than fifty million adult population in the world would be affected with cirrhosis liver.[2,3]

In the Western world alcoholic liver disease and hepatitis C remains the most common cause of cirrhosis, while hepatitis B prevails in most parts

of Asia and sub-Saharan Africa.[4] Currently along with alcohol and viral hepatitis, nonalcoholic steatohepatitis (NASH) is another most common etiology for cirrhosis.2 Biological membranes, free molecules and metabolic regulators contains lipid and are essential in controlling cellular function and homeostasis. Liver has important role in lipid metabolism hence it is profoundly disturbed in a variety of ways in severe liver disease.[5]

Circulating lipoproteins are seen in abnormal amount, abnormal composition, electrophoretic mobility and appearance.[5]Cardiovascular disease (CVD) risk stratification includes serum lipid profile. It is rarely considered a useful screening tool for the evaluation of liver diseases, yet there is reason to think otherwise.[6] American Heart Association guideline, 2013 suggest once therapy for dyslipidemia is started there is no need of monitoring.[7] Many previous studies concluded an inverse association of lipid parameters such as total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) with severity of liver disease. However, some other studies did not find such correlation especially for the TG and HDL levels.[8,9] Further, Sen et al showed that TC, HDL and TG were higher in grade 3 fatty liver.[10] This warrant the significance of a population based evaluation of the lipid profile in subjects with liver disease. Hence, this study was conducted to find out the correlation of lipid profile in patients with severe liver disease.

Operational definition

- **Chronic Liver Disease:** CLD was diagnosed on ultrasonography with small size liver (size<13 cm) having coarse texture liver and having one of the following in addition:
  - Portal vein diameter >10mm.
  - Splenomegaly: size of spleen (length)> 13 cm on ultrasound.
  - Ascites: shifting dullness +ive and confirmed on ultrasound.

MATERIAL AND METHODS

This cross sectional study was conducted at Department of Gastroenterology & Hepatology, Bahawal Victoria Hospital, Bahawalpur from February 2018 to August 2018 over the period of 6 months.

Total 123 patients with age ≥18years either male or female with chronic liver disease or documented case of chronic liver disease irrespective of aetiology were included in the study. Proven cases of dyslipidemia prior to detection of chronic liver disease, recent parenteral nutrition, patient on immunosuppressive drug, patients with BMI >30 or patients unwilling to participate in the study were excluded. Informed consent was obtained from either the patient or bystander. The study was conducted after acquiring consent from the institutional scientific and ethics committee.

Detailed history was taken from each patient to ascertain past and present illness. All the patients were subjected to a thorough physical examination using specific proforma. Severity of liver disease was calculated according to Child Pugh Turcotte Score.Patients were subjected to routine investigation and fasting lipid profile test. Routine test included complete blood count (CBC), urine routine, random blood sugar, renal function test, liver function test, HBsAg, HCV antibody, Prothrombin time (PT/INR), ultrasonography of whole abdomen. A fasting serum lipid profile included TC, TG, HDL and LDL.

The data were analyzed using the SPSS software. Correlation was studied using the Pearson correlation coefficient. Comparison of lipid parameters with the severity of liver disease was done using Kruskal-Wallis Test. $P<0.05$ was considered significant.

RESULTS

Mean age of the patients was 38.01 ± 14.68 years. Out of 123 patients of chronic liver disease, male patients were 104 (85%) and female patients were 19 (15%). (Fig. 1)

Patients were divided into 6 age group i.e. age group <30 years, age group 31-40 years, age group 41-50 years, age group 51-60 years, age group 61-70 years and age group >70 years. In age group <30 years, there were 1 (0.08%) patients, followed by 7 (5.7%) in age group 31-40 years group, 25 (20.3%) in 41-50 years group, 49 (39.8%) in 51-60 years, 32 (26%) in age group 61-70 years and 9 (7.3%) in age group >70 years group. (Fig. 2)

Total 24 (20%), 47 (38%) and 52 (42%) patients belonged child pugh class A, B and C. (Fig. 3)

In childpugh class A, B and C, Mean Total cholesterol was 139.08±40.16 mg/dl, 141.30±51.54 mg/dl and 105.81±40.26 mg/dl respectively. Mean LDL was 84.29±35.16 mg/dl, 88.55±48.13 mg/dl, 76.67±79.19 mg/dl respectively in child pugh class A, B and C. Mean HDL was 34.42±14.67 mg/dl, 29.81±12.82 mg/dl and 21.23±11.73 mg/dl in child pugh class A, B and C. Mean TG was 109.17±64.04 mg/dl, 116.32±54.99 mg/dl and 90.06±53.35 mg/dl respectively in child pugh class A, B and C. (Table 1) As shown in table 2, negative correlation between lipid profile and child pugh class was noted.

Fig. 1 Gender distribution

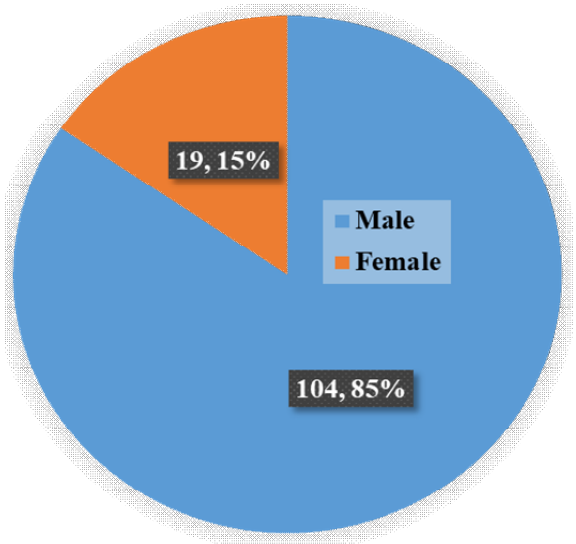

Fig. 2: Age distribution

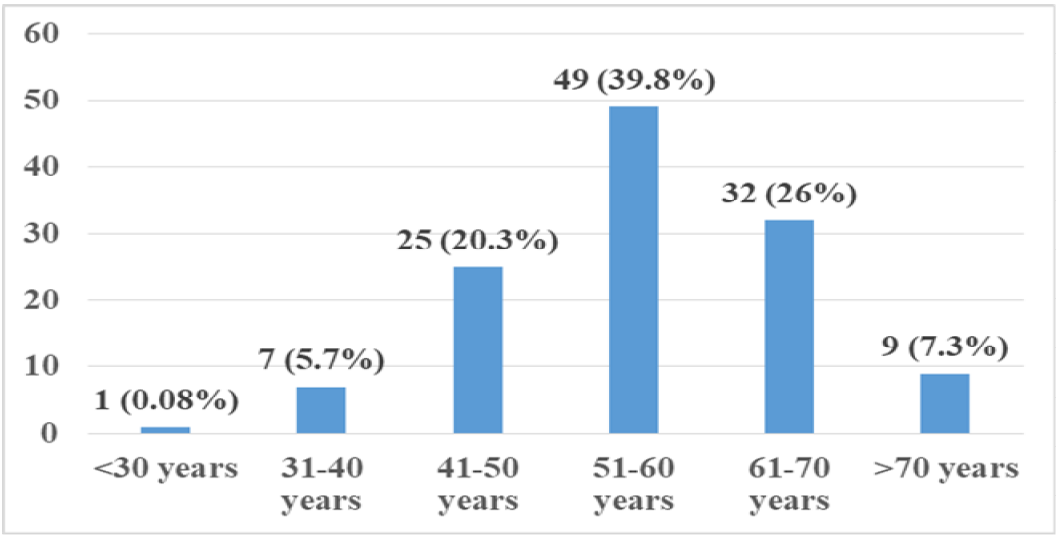

Fig. 3: Child pugh class distribution

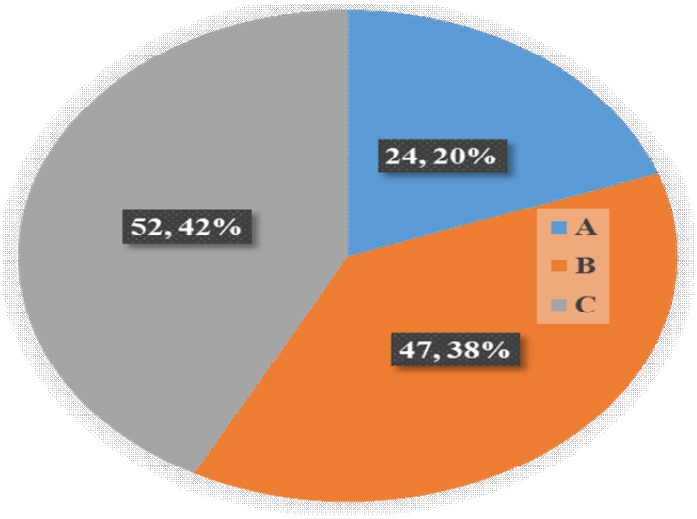

Table 1: Serum lipid profile in patients with chronic liver disease.

Lipid Profile	Child Pugh Class			P value
	A	B	C	
Total cholesterol (mg/dl)	139.08±40.16	141.30±51.54	105.81±40.26	0.0001
LDL (mg/dl)	84.29±35.16	88.55±48.13	76.67±79.19	0.013*
HDL (mg/dl)	34.42±14.67	29.81±12.82	21.23±11.73	0.0001*
TG (mg/dl)	109.17±64.04	116.32±54.99	90.06±53.35	0.004*

Table 2: Correlation between the laboratory parameters with the severity of disease.

Parameter	HB	MCV	PLT	PT INR	BIL-T	ALB	S CHL	LDL	HDL	TG
R value	-0.130	-0.018	-0.349	0.691	0.661	-0.706	-0.387	-0.129	-0.434	-0.191
P value	0.152	0.840	0.0001	0.0001	0.0001	0.0001	0.0001	0.154	0.0001	0.034

HB: hemoglobin; MCV: Mean corpuscular volume; PT/INR: Prothrombin time in international normalized ratio; BIL-T: Total bilirubin; ALB: Albumin; S CHL: Serum cholesterol; LDL: Low density cholesterol; HDL: High density cholesterol; TG: Triacyl glycerol.

DISCUSSION

Lipids are one of the necessary components which control cellular functions and homeostasis. Liver plays an essential role in lipid metabolism, several stages of lipid synthesis and transportation. Therefore, it is reasonable to expect an abnormal lipid profile in those with sever liver dysfunction. There is prominent decline in plasma cholesterol and triglyceride (TG) levels in patients with severe hepatitis and hepatic failure because of reduction of lipoprotein biosynthesis. For reduced liver biosynthesis capacity, low levels of TG and cholesterol is usually observed in chronic liver diseases.[4,8]

The result of the present study revealed that more number of patient were in class C. All the parameters of lipid profile were lower in the severe form of liver disease irrespective of the etiologies. Furthermore, the amount of decrement in the serum HDL, LDL, TC and TG had a negative correlation with the severity of liver disease. This indicated an inverse correlation of lipid parameters with the severity of the disease. The result was consistent to the previous studies.[5,11-13] They found a reduction in all parameters of lipid profile with severity of chronic liver disease. However, there is conflicting of

observations on this regard. Previous study by Mandal et al found thatserum TG and HDL levelwere not reduced with severity of liver cirrhosis.8

The decreased LDL and HDL levels found in patients with liver cirrhosis might be ascribed to the decreased synthesis of apolipoproteins (Apo) A and B. Habib et al previously reported that decline in lipoprotein cholesterol may reflect deterioration lipoproteins synthesis in liver.[14,15] Since the apo B is involved in the synthesis of very low density lipoproteins, the exhibited lowering of TG can be explained to its lowered synthesis in liver. This can be due to the insulin resistance (IR) found in liver diseases. Insulin signalling mechanism was found to be critical for the lipogenesis in hepatocytes by regulating the PI3K and AKT2 signalling pathways.[16,17] Among the transcription factors, sterol regulatory element binding protein-1c (SREBP-1c) has stimulatory effects on the expression of genes involved in lipogenesis.[18] Insulin was found to stimulate the lipogenesis via activation SREBP-1c.18 Therefore, the decrease in lipid parameters can be ascribed to the IR which possibly more in subjects with cirrhosis.[19] During IR states, AKT2 was involved in the hepatic lipid accumulation, explains one of the etiological factor for lipid accumulation in liver.[17] The patho-physiology of fatty liver disease was initiated from the deposited fat in hepatocytes followed by the inflammation and oxidative stress.[16] As the disease advances to liver fibrosis and cirrhosis, inflammation and oxidative stress were involved in the augmentation of the IR.

CONCLUSION

Results of present study showed a higher percentage of male patients as compared female suffering with chronic liver disease. Most of the patients belonged to age group 51-60 years. Negative correlation between lipid profile and child pugh class was found.

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